The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 6, 2025

EFFECT OF DIFFERENT DIALYSATE SODIUM CONCENTRATIONS ON BLOOD PRESSURE RESPONSE DURING HEMODIALYSIS

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DOI: https://doi.org/10.5281/zenodo.19219018

Keywords

Article History
Received: 12 May 2025
Accepted: 05 June 2025
Published: 21 June 2025

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Abstract

Objective:
The objective of this study is to observe effect of different dialysate sodium concentrations on blood pressure response during hemodialysis

Study design:
Randomized Control trial study.

Place and duration of study:
Department of Nephrology, Tertiary Care Hospital, Lahore from February 2025-April 2025.

Methodology:
105 patients undergoing hemodialysis were inducted in the study to assess the effect of different dialysate sodium concentrations on blood pressure response during maintenance hemodialysis, ESRD patients on thrice-weekly hemodialysis were enrolled. Participants underwent dialysis with sodium concentrations of 135, 138, and 142 mmol/L in randomized sequences, each for two weeks with washout periods. Blood pressure was measured pre-, intra-, and post-dialysis. The primary outcome was change in systolic blood pressure. Data were analyzed using repeated-measures ANOVA, with $p < 0.05$ considered significant.

Results:
A total of 105 patients (mean age 52.6 ± 13.4 years; 58.1% males) completed the study. Hypertension, diabetes, and cardiovascular disease were present in 82.9%, 46.7%, and 28.6% of participants, respectively. Lower dialysate sodium (135 mmol/L) resulted in the greatest intradialytic reductions in systolic and diastolic blood pressure and the highest rate of hypotension. Higher sodium (142 mmol/L) provided superior hemodynamic stability. The standard concentration (138 mmol/L) showed a balanced profile. Repeated-measures ANOVA demonstrated statistically significant differences across sodium levels for most primary and secondary outcomes ($p < 0.001$).

Conclusion:
Dialysate sodium concentration significantly affects intradialytic hemodynamic stability and interdialytic cardiovascular outcomes in maintenance hemodialysis patients. Lower sodium improves predialysis blood pressure control but increases the risk of intradialytic hypotension.

INTRODUCTION:

Hemodialysis is a life-sustaining therapy of the patients with the end-stage renal disease, but intradialytic blood pressure instability is a common complication that can influence the morbidity, mortality, and quality of life. The dialysate sodium level is important to define hemodynamic reactions during dialysis as it affects the plasma osmolality, fluid changes, vascular tone and thirst mechanisms. The modification of dialysate sodium is thus seen to be a modifiable variable that can maximise the stabilisation of blood pressure and lead to better outcomes in the patients.[1]

An increase in dialysate sodium levels is usually linked with an increase in intradialytic hemodynamic tolerance. Maintaining plasma osmolality, it is due to high sodium concentrations that osmotic fluid moves between intravascular and intracellular space reduce rapid changes in circulating volume. This process reduces cases of intradialytic hypotension, muscle cramps as well as dizziness. Nevertheless, long-term exposure to high levels of dialysate sodium can cause an elevation in interdialytic weight gain, high blood pressure, and increased cardiovascular risk because of increased thirst and fluid consumption.[2]

Reduced dialysate sodium levels on the other hand can be used to manage predialysis hypertension and interdialytic weight gain by limiting sodium loading. Research indicates that low-sodium dialysate could have a beneficial effect on the long-term blood pressure regulation and lower the need of the antihypertensive medication. However, too low levels may predetermine patients to intradialytic hypotension and other symptoms especially in patients with autonomic dysfunction, heart conditions, or large ultrafiltration needs.[3]

One-on-one prescriptions of sodium have thus become a promising approach. Personalization of dialysate sodium according to a serum sodium level of a patient can provide a balance between acute hemodynamic stability and cardiovascular safety in the long run. In modern dialysis machines, sodium profiling is also possible whereby elevated sodium is administered at the beginning of the therapy and then decreased, which may potentially reduce hypotension and sodium overload.[4]

The results in conclusion indicate that dialysoodium is a significant factor in blood pressure reactions during hemodialysis. Increased concentrations enhance intradialytic stability at the expense of chronic hypertension and fluid overloading, and low concentrations enhance long-term blood pressure management at the expense of acute hypotensive crises. Individual dialysate sodium prescriptions grounded on patient characteristics, comorbidities and treatment objectives are the most reasonable way of optimizing hemodynamic outcomes.

Methodology:

The research was done in the form of a prospective, randomized, controlled, trial that aimed at testing the impact of the various dialysate sodium levels on the blood pressure response to the maintenance hemodialysis. The trial was conducted in hemodialysis unit/Nephrology department of a tertiary care hospital, Lahore during a period of 03 months, between February 2025 and April 2025. The institutional review board approved the ethical approval and informed consent will be acquired through written consent of all the participants before enrollment. Eligible patients were adult patients with end-stage renal disease under maintenance hemodialysis three times in a week and at least three months. Consecutively, patients with inclusion criteria were enrolled until the target sample size was attained. The power analysis which was based on previous studies that compared intradialytic blood pressure changes to varying dialysate sodium levels was used to compute the sample size. Given a level of significance of 0.05, 80 power and a moderate effect size of systolic blood pressure difference between interventions to be expected, the calculated sample size was 105. The randomization was carried out through computer-generated randomization whereby participants would be assigned to various dialysate sodium sequences. All patients were also subjected to dialysis through at least three sodium concentrations (such as low 135 mmol/L, standard 138 mmol/L and high 142 mmol/L), each with a fixed period of time (such as

two weeks), with washout periods between them using standard sodium dialysate. The crossover design will provide both participants with their own control and inter-individual variability was minimised. Dialysis time, dialyzer, blood flow rate, dialysate flow rate, temperature and ultrafiltration targets of each participant will remain constant during the period of the study so as to reduce confounding factors.

Baseline demographic and clinical information involved age, sex, dialysis vintage, cause of renal failure, comorbidities, antihypertensive medication use and baseline laboratory values. An automated sphygmomanometer was performed to measure blood pressure before the dialysis, at 30-minute intervals during the dialysis, and after dialysis. Systolic blood pressure was altered as the primary outcome in the process of dialysis. The secondary outcomes included change in diastolic blood pressure, intradialytic hypotension episodes, muscle cramps, dizziness, saline intervention, interdialytic weight gain and post-dialysis recovery time.

Inclusion Criteria

1. Adults aged ≥18 years
2. Diagnosed with stage 5 renal disease.
3. Maintenance hemodialysis 3 months or more.
4. No hospitalization or stable clinical status in past 4 weeks.
5. Capability to make informed consent.

Exclusion Criteria

1. Unstable cardiovascular (recent myocardial infarction, unstable angina, severe arrhythmias)
2. Active infection or sepsis
3. Serious liver disease or malignancy with poor prognosis to live.
4. Pregnancy
5. Individualized sodium prior to study, patients under sodium profiling dialysis, or individualized sodium prescription.
6. Failure to adhere to dialysis or drug schedule.
7. Failure to handle protocol sodium levels.

The main outcome were the mean intradialytic change of systolic blood pressure with varying sodium concentrations. Secondary outcome measures are incidence of intradialytic hypotension, symptomatic events, interdialytic weight gain and need of clinical interventions.

The statistical software (SPSS, R, etc.) was used to analyze the data: continuous variables was indicated as the mean, standard deviation; categorical variables was indicated as frequencies and percentages. To compare the responses of blood pressure to various sodium levels, repeated-measures ANOVA or mixed-effects modeling wias were considered. Bonferonni-adjusted post hoc pair-sample comparisons were found to identify specific group differences. The p-value used as a significant value was less than 0.05.

Results:

One hundred and five patients went through the research. The average age was 52.6±13.4 years and the number of males was 61 (58.1%) and 44 females (41.9%). Mean dialysis vintage was 4.2 ±2.1 years. Hypertension, diabetes mellitus and cardiovascular disease were found in 82.9, 46.7 and 28.6 percent of participants respectively. The exposure to three levels of dialysate sodium concentration (135, 138, and 142 mmol/L) was accomplished by the participants; no protocol violations were documented.

Greater concentrations of dialysate sodium were linked to substantially lesser decreases in the systolic and diastolic blood pressure.

Increase in sodium dialysate lead to a large increase in interdialytic weight change, thirst and predialysis systolic blood pressure.

Summary of Key Findings:

- The intradialytic decrease in blood pressure was maximum and the rate of hypotension was the highest with lower dialysate sodium (135 mmol/L).
- Higher sodium (142mmol/L) was associated with the greatest hemodynamic stability, but an increased interdialytic weight gain and predialysis hypertension.

- St. Na+ (138 mmol/L) provided a balanced profile, moderate stability, and less long-term adverse effects.
- The ANOVA of repeated-measures revealed that all primary and secondary outcomes were statistically significant ($p < 0.001$ in most comparisons) due to dialysate sodium concentration.

Table 1. Baseline Demographic and Clinical Characteristics

Variable	Value
Age (years), mean ± SD	52.6 ± 13.4
Male, n (%)	61 (58.1%)
Dialysis vintage (years)	4.2 ± 2.1
Diabetes mellitus	49 (46.7%)
Hypertension	87 (82.9%)
Cardiovascular disease	30 (28.6%)
Baseline serum sodium (mmol/L)	137.8 ± 2.9
Antihypertensive drugs (mean no.)	1.9 ± 0.8

Table 2. Blood Pressure Response Across Dialysate Sodium Levels

Parameter	135 mmol/L	138 mmol/L	142 mmol/L	p-value
Pre-dialysis SBP (mmHg)	151.2 ± 18.4	150.6 ± 17.9	150.9 ± 18.1	0.94
Post-dialysis SBP	126.3 ± 16.2	132.8 ± 15.4	138.7 ± 14.9	<0.001
Mean SBP drop	−24.9 ± 10.5	−17.8 ± 9.2	−12.2 ± 8.8	<0.001
Mean DBP drop	−11.6 ± 6.1	−8.9 ± 5.8	−6.4 ± 5.1	<0.001

Table 3. Intradialytic Hypotension Episodes

Sodium Level	Episodes (n)	% Sessions Affected
135 mmol/L	64	20.3%
138 mmol/L	38	12.1%
142 mmol/L	21	6.7%

Table 4. Interdialytic Outcomes

Parameter	135 mmol/L	138 mmol/L	142 mmol/L	p-value
Interdialytic weight gain (kg)	2.1 ± 0.6	2.5 ± 0.7	3.0 ± 0.8	<0.001
Thirst score (Likert 1–5)	2.1 ± 0.9	2.9 ± 1.0	3.8 ± 0.8	<0.001
Pre-dialysis SBP	146.7 ± 16.5	150.6 ± 17.9	156.4 ± 18.2	<0.001

Discussion:

The findings of this research give a good substantiation that dialysate sodium level is a strong predictor of intradialytic hemodynamic reactions in maintenance hemodialysis patients. Lower dialysate sodium (135 mmol/L) was found to be coupled with increased reductions in systolic and diastolic blood pressure during dialysis, increased incidences of intradialytic hypotension, and increased intradialytic symptoms (muscle cramps and dizziness) in our 105-patient cohort. On the other hand, increased dialysate sodium (142 mmol/L) increased blood pressure during dialysis but also increased interdialytic weight gain and thirst scores, and predialysis blood pressure.

The normal sodium concentration (138 mmol/L) showed moderate results. These results are in line with other known data, but also offer a sense of specificity that can be applied in short-term and even longer clinical management.

Other clinical studies have also indicated that dialysate sodium levels do have substantial influences on blood pressure patterns during the process of hemodialysis. Low sodium (5 mmol/L below serum sodium) in a randomized crossover trial by Inrig et al., significantly reduced systolic blood pressure during treatments compared to high sodium (5 mmol/L above serum sodium), which supports the idea that the lower the sodium, the better is intradialytic blood pressure control. In the same way a recent prospective study showed that ambulatory blood pressure during interdialytic period of 72 hours progressively increased with higher dialysate sodium levels (137 139 141 mmol/L) which indicated that sodium not only influenced immediate hemodynamics but also extended blood pressure profiles beyond the dialysis unit. These observations are compatible with our observations that indicate an increase in dialysate sodium can play a role in the continuous increase in systemic blood pressure.[5,6]

Dialysate sodium affects fluid shifts and plasma osmolality in a mechanistic manner. The high dialysate sodium will aim to keep the circulating volume constant by decreasing fluid loads into the intracellular space contributing to reduced incidences of intradialytic hypotension, but may also cause sodium loading and thirst, and result in greater interdialytic weight gain. This opportunity cost was apparent in our experiment: increased interdialytic weight gain and thirst score were observed in bigger dialysate sodium which have the potential to increase long-term hypertension and cardiovascular astuteness.[7]

There is contradictory evidence. In dialysis patients in the hospital, a large randomized trial demonstrated no significant difference in intradialytic blood pressure fall or in the incidence of hypotension between dialysate sodium 142mmol/L and 138mmol/L. These findings indicate a possibility that the effect of sodium concentration on hemodynamics could be mediated by patient population, setting (ambulatory vs hospitalized) and comorbid conditions.[8]

Our findings are also supported by longitudinal studies and multicenter observational studies. High dialysate sodium (>140 mmol/L) in PanThames Renal Audit demonstrated greater weight gains interdialytic and increased pulse pressure whereas low sodium (136137 mmol/L) was associated with reduced weight gains and reduced symptomatic hypotension. A survey conducted on chronic dialysis centers revealed that centers with a higher dialysate sodium concentration had less blood pressure control and more patients were requiring antihypertensive pharmaceuticals. These cohort results have a resonance in our observations that dialysate sodium has a systemic impact beyond the individual program of dialysis.[9,10]

This complexity can be observed in international guidelines and the opinion of the experts when it comes to dialysate sodium prescription. Conventionally, a standard dialysate sodium level of about 138-140 mmol/L has been in use and this is based on average levels of serum sodium in dialysis patients. Recent professional articles, however, warn that dialysate sodium should not be used without specifications and instead should be personalized depending on patient fluid condition, symptoms, and cardiovascular risk. As an example, dialysate sodium reduction can help patients with inadequate hypertension management as well as surplus interdialytic weight gain, but only with the close observation of intradialytic symptoms. On the contrary, a small additive of dialysate sodium or sodium profiling can be beneficial in patients with recurring hypotension or possessing a severe amount of cardiac disease.[11]

It is also the results of ours which highlight the necessity of balanced clinical judgment. Although the use of lower sodium enhanced the overall blood pressure control, it was at the expense of the increase in the frequency of the hypotensive episodes and intradialytic symptoms. Such acute complications may affect comfort and safety of the patients, especially those with autonomic dysfunction or high ultrafiltration needs. Sodium profiling or tailored prescriptions with

consideration of pre-dialysis serum sodium levels have been suggested as a standardized or personalized method of establishing optimal interaction between intradialytic stability and sodium load during the long-term, but these are early studies.

The results of the current research also reinforce the interrelationship between sodium regulation and extracellular volume as well as vascular responsiveness in support of hemodialysis, which is highly intricate physiologically. Instead of being an electrolyte that is lost or replaced during dialysis, sodium is a primary predictor of osmotic balance and neurohormonal stimulation. Changes in dialysate sodium concentration have effects on plasma osmolality that direct water transport across the cell membrane, the volume of circulating blood and the resistance to vascularization. Positive sodium gradient forms when dialysate sodium is greater than serum sodium resulting in diffusion of sodium into the patient and maintenance of plasma osmolality. This dampens the effect of the acute contraction of intravascular volume during ultrafiltration and decreases the potential of symptomatic hypotension. On the other hand, in case dialysate sodium is less than serum sodium, increased sodium loss lowers extracellular volume at the expense of sudden intravascular loss and hemodynamic variability.[12]

These physiological findings are backed by a number of international studies. The Dialysis Outcomes and Practice Patterns Study (DOPPS) indicated that there is a great divergence in dialysate sodium prescriptions across countries with higher concentrations generally being prescribed to patients in North America than in some parts of Europe and Asia. Notably, greater dialysate sodium was related to increased interdialytic weight gain and increased predialysis systolic blood pressure in observational studies.These findings are similar to the results of our study, where an increased concentration of sodium (142 mmol/L) was related to significantly higher levels of interdialytic weight gain and predialysis systolic blood pressure.[13]

In the same manner, Hecking et al. analyzed the DOPPS data and stated that higher dialysate sodium levels than serum sodium levels in patients were correlated with higher fluid intake and weight gain, without demonstrable long-term survival advantage. Prolonged extracellular volume increase has been found to induce left ventricular hypertrophy, arterial stiffening, and endothelial dysfunction, which are cause of significant cardiovascular death in patients with end-stage renal disease.[14]

The same has been found by national data on the South Asian dialysis population, which includes observational research data in tertiary centers in Pakistan and India, which indicates that routine dialysate sodium levels of 140 mmol/L or higher were also linked to poor blood pressure control and increased antihypertensive medication needs. These results are similar to our local data, which supports the significance of local research. Disparities in dietary sodium consumption, fluid intake in response to climate conditions, patient body structures could also affect how sodium is managed in the locals abroad and thus national results should be combined with international results.[15]

Meanwhile, there is nothing riskless about aggressive sodium restriction. Flythe et al. established that intradialytic hypotension is a significantly independent predictor of excess mortality, especially recurrent and severe hypotension, creating the issue of balancing between the acute safety and the chronic blood pressure management. Repeated subclinical myocardial ischemia, cerebral hypoperfusion, and vascular access thrombosis can be brought about by intradialytic hypotension. Thus, interventions that promote the reduction of predialysis blood pressure by means of sodium limitation should take into account the possible damage of regular hypotensive episodes.[16]

New data is becoming available that the idea of patient specific sodium set point may be the cause of variability in response. According to Mc Causland et al., the agreement between dialysate sodium and personal serum sodium level decreased sodium flux and enhanced the hemodynamic condition without excessive weight gain. This idea is indirectly confirmed in our results because intermediate sodium (138

mmol/L) was found to give a more balanced result profile than extreme concentrations.[17]

Compromise strategies have also been investigated such as sodium profiling whereby dialysate sodium is initially set at a higher level and progressively set at a lower level throughout treatment. Song et al showed that sodium modeling, together with ultrafiltration profiling, lowered symptomatic hypotension but did not raise interdialytic weight gain significantly, but there is still the concern that sodium profiling can still lead to net positive sodium balance unless closely monitored. There is a dearth of long term cardiovascular outcomes of sodium profiling.[18]

The other dimension is that of arterial stiffness and endothelial functioning. Inrig et al. noted that reduced dialysate sodium enhanced endothelial-dependent vasodilation, which may be cardiovascular beneficial in addition to blood pressure lowering. Though our study did not determine the vascular biomarkers, the better predialysis blood pressure may be due in part by better vascular tone with time.

Pathophysiologically, dialysis patients do not entirely depend on dialysate prescription to define sodium balance. There is dynamic interaction between residual renal functioning, dietary sodium consumption, dialysate temperature, ultrafiltration rate, and compliance of patients with fluid restriction. There is a multifactorial relationship between dialysate sodium and blood pressure. Further research combining bioimpedance analysis, ambulatory blood pressure measurements and cardiac imaging could offer to deliver a more in-depth assessment of the long-term cardiovascular outcomes.

Notably, our results contribute to the ever-expanding literature recommending dialysis treatment that is individualized. Standardized dialysate sodium orders do not reflect the heterogeneity of cardiovascular status, autonomic functioning, and sodium sensitivity. Marginal sodium or sodium profiling can be useful to patients who are likely to experience recurrent hypotension, though those with refractory hypertension and excessive weight gain may need to undergo careful sodium intake with close monitoring.[19]

Summing up, the detailed discussion proves that dialysate sodium level has a lot of impact on immediate intradialytic hemodynamics and overall cardiovascular parameters. The recommended sodium dose seems to be in a patient-based, tailored treatment that harmoniously considers acute stability and long-term heart safety. There is a need to conduct large, multicentered, randomized trials with a long period of follow up to establish the effects of personalized sodium interventions on hard clinical outcomes like hospitalization, cardiovascular events, and death.

Limitation of study:

In spite of the strength of our study due to its crossover design and immense sample size, further longitudinal studies are required to determine the effects of dialysate sodium modification on cardiovascular incidents and mortality. Also, individualized sodium prescriptions may be refined by investigating the importance of patient-based variables (residual renal function, sodium dietary intake, and antihypertensive treatments).

Conclusion:

This research indicates that the concentration of Na in the dialysate is a key-factor that influences the intradialytic stability of the hemodynamic in patients undergoing maintenance hemodialysis and interdialytic cardiovascular parameters. Reduced dialysate sodium also caused larger changes in systolic and diastolic blood pressure and better control of predialysis blood pressure, which indicates its applicability in patients with intractable hypertension or excessive fluid retention. The usual concentration of sodium) exhibited a moderate threshold of the chronic burden of cardiovascular and the acute stability profile. These results support the idea that dialysate sodium prescription cannot be the same across the patients but should rather be tailored according to the clinical features, blood pressure fluctuations, and tolerance. Individual sodium plans, possibly along with sodium profiling and close observation, might maximize patient comfort, cardiovascular stability and long-term

outcomes in maintenance hemodialysis populations.

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